NavMed P-369

UNITED STATES NAVY

MEDICAL NEWS LETTER

Vol. 37 Friday, 17 March 1961 No. 6

TABLE OF CONTENTS

United States Navy

MEDICAL NEWS LETTER

Vol. 37 Friday, 17 March 1961 No. 6

Policy

The U. S. Navy Medical News Letter is basically an official Medical Department publication inviting the attention of officers of the Medical Department of the Regular Navy and Naval Reserve to timely up-to-date items of official and professional interest relative to medicine, dentistry, and allied sciences. The amount of information used is only that necessary to inform adequately officers of the Medical Department of the existence and source of such information. The items used are neither intended to be, nor are they, susceptible to use by any officer as a substitute for any item or article in its original form. All readers of the News Letter are urged to obtain the original of those items of particular interest to the individual.

* * * * * *

Change of Address

Please forward changes of address for the News Letter to: Commanding Officer, U. S. Naval Medical School, National Naval Medical Center, Bethesda 14, Md., giving full name, rank, corps, and old and new addresses.

* * * * * *

Use of funds for printing this publication has been approved by the Director of the Bureau of the Budget (19 June 1958).

The Drug Explosion

Walter Modell MD, Director, Clinical Pharmacology, Cornell University Medical College, New York City. Editorial, Clinical Pharmacology and Therapeutics 2: 1-7, January - February 1961.

If the pharmaceutical chemists took time to look back at the net result of their prolificacy, would they be shocked to discover that the point of no return may have been passed? Do they suspect that now, instead of helping mankind with new drugs, they may be making matters worse? Although no one would suggest that they cease or even slow the pace of their search for useful drugs, if they are at all interested, I suggest that they do take the time to follow the effect of their creativity to its ultimate conclusion.

Even 5 years ago, it was pointed out that to the already staggering total of about 140,000 medicaments in current use—of which an estimated 90% did not exist 25 years before and 75% had been introduced within 10 years—some 14,000 new ones had been added during the current year. Untoward reactions to medication have also increased at a staggering rate. This comes about primarily because of inability to master the full implications of these agents as rapidly as they are marketed. Of much less importance are the unpredictable and unavoidable cases of hypersensitivity which are relatively rare.

As the number of new and active drugs increases and both knowledge and experience with each therefore become smaller and proportionately more difficult to obtain, it is a mathematic inevitability that drug reactions will also increase. Furthermore, since more than a single factor is involved, it is also certain that the incidence of reactions will increase even faster than the rate at which new drugs emerge. Even now the situation is alarming, but the future looks dismal indeed.

If this situation were a hazard inherent in medical progress there would be some justification for it, but too often this is not the case. Too often drugs are turned loose on the market strictly for business reasons. Thus, drugs are marketed, promoted, and advertised by precisely the same technics as those used for soaps and detergents.

A large proportion of claims for superiority for new drugs is patently invalid; it is impossible that, of the huge number of new drugs available, each one is the "best" for a separate medical indication. Of course, there would be no confusion if the pharmaceutical industry saw the lack of ethics in claiming a drug to be the best when a better drug was, in fact, available, and if the sole criterion for introduction of a drug was the good of the patient. At the moment, the most helpful contribution is the new drug to counteract the untoward effects of other new drugs.

Of the new drugs, some are extremely potent and exert diffuse effects, others interfere with basic physiologic function; many are most unusual pharmacologically, hence poorly understood; many suffer from limited clinical trial;

all are vigorously advertised. Too many are used with little discrimination. Dr. Wrightman stated that if ever we were in danger of irrational behavior as therapeutists, it is now.

Are physicians characteristically irrational and irresponsible? No, but they may sometimes appear to be because of the sheer impossibility of dealing rationally and responsibly with so many new drugs about which so little is known, but for which extravagant claims are made, and for use of which pressure is exerted by the drug industry and by patients who have heard of new cures through newspapers, magazines, and other patients. It is beginning to look as if the success of a new drug will depend less on how well it works and more on how well it is promoted.

That the situation gives every indication of worsening is suggested by the title of a paper on steroids, "How to Win at Structural Roulette." The pharmaceutical industry has had a prolonged winning streak at this game, but every winning streak ends some time. What will happen when physicians refuse to gamble with their patients' lives and health, or an enraged public demands that such gambling stop? The reaction may lead to unhealthy cynicism and a state of exaggerated therapeutic nihilism on the part of physicians.

If the pendulum then swings as far in the other direction—as pendulums do—the medical profession will tend to lean on a handful of older proved drugs. In this counterplay, surely important discoveries of our time will be overlooked and lost. How long before the public, the medical profession, and the drug industry are the losers to this type of general reaction?

If the therapeutic morbidity continues to rise—as it must under present conditions—it is clear that something will have to be done. Is governmental control the only answer? The answer lies within the drug industry. The industry has done many wonderful things for medicine and for mankind. It is to be hoped that it will continue to do so; it has a moral obligation to the community which is in no wise lessened by its contributions of the past.

What can the pharmaceutical industry do? Industry should undertake to control its practices. It should plan broadly for effective screening of the drugs it synthesizes and should terminate the practice of hurried introduction of new drugs in order to establish a foothold on the market while leaving the real testing of drugs in the hands of practicing physicians. Industry should undertake to limit the number of congeners of a single drug on the market to some practical number. If clinical trials were carried out on a grand scale with an entire group of drugs examined in a coordinated program at one time, the truth about the group as a whole as well as the relative merits of its members would emerge much more promptly.

Can there be any doubt that under such a system the public and the medical profession would benefit? If the medical profession merely continues to use drugs when they are needed, it will obviously not prescribe less after the inauguration of such a system than it does now. It is possible that it might prescribe even more because it would feel more secure. There might well be

fewer token prescriptions given because of patient demands for the latest in drug development.

Would not discovery of new drugs pay off even better than now, since new drugs would be more firmly established by this system? Because their effective lives would be longer, their use through cross-licensing would be more extensive and the income to the discoverer through royalties should be greater. In addition, "the most ethical of the ethical companies" will not be forced by competition—as the New England Journal of Medicine points out they now are—to "meet the tactics of the least ethical."

Under restrictions, such as suggested, research in industry would continue at its present pace; new drugs will always be needed. But under such a system, those drugs which reach the market would be used without reluctance and cynicism because faith in industry's claims for drugs would be reestablished.

There can be no question of the fact that the medical profession's gain would be enormous. Because there would be fewer drugs in use, there would be a much larger over-all experience with each and more information about the unusual as well as the more common drug actions. As the result of more knowledge and greater confidence, the physician would use his medicaments with greater assurance and exploit drugs to the greater good of his patients.

Would the patient benefit? Only the best drugs would be available. Because the physician would understand the drugs better and use them more effectively and with greater confidence, patients would get better results from them. Certainly there would be far less iatrogenic disease. And with a stable and more prolonged market, drug costs to the patient would be less, yet with an increase of profits to the manufacturer.

It could happen here.

* * * * * *

The Rubella Problem

Clifford W. Skinner Jr, MD, Department of Pediatrics, University of Colorado School of Medicine, Denver, Colo. Amer J Dis Child 101: 78-86, January 1961.

Rubella was considered a relatively unimportant disease of childhood until 1941, when Gregg in Australia reported congenital cataracts in offspring of women who had contracted the disease while pregnant. Since that time, numerous other congenital defects as well as complications of pregnancy have been documented. These events which follow maternal rubella have become known as the rubella syndrome.

Classification of Rubella Syndrome. The major components of the rubella syndrome are the congenital defects occurring alone or in combination,

and complications of pregnancy. The former consist of eye defects, congenital heart disease, dental defects, microcephaly, mental retardation, deafness, and failure to thrive; the latter include spontaneous abortions and stillbirths. Other congenital lesions have been noted to occur following rubella, but their association with the disease has yet to be proven. These include hypospadias, biliary atresia, mongolism, talipes equinovarus, syndactylism, cleft palate, and generalized muscular weakness.

Incidence. Accurate statistics are difficult to collect. Because of the mild nature of the disease, many cases are not reported. In 1951 during an epidemic, 1067 cases of maternal rubella occurring during pregnancy were reviewed: 17% of pregnancies in which rubella was present during the first 4 months resulted in stillbirths, neonatal deaths, immaturity, or congenital anomalies. When neonatal deaths and stillbirths were eliminated from the group, 9 to 12% of surviving infants had congenital anomalies. More recent figures agree with this study.

Observations during this epidemic suggest that previous history of the disease is not sufficient to protect the fetus; later reports support this impression. In Ireland, one group of women who were exposed to, but did not develop, rubella produced offspring with anomalies at a rate that was 3.5 times as great as seen in the general population. This consequence may be due to a possible mechanism of viremia developing in the mother, although her immunity eventually overcomes the invasion without development of disease. However, passage of the virus to the fetus may not be prevented and be in sufficient degree to affect development.

Clinical Findings. In the U.S. there is a seasonal variation of occurrence of rubella with a peak from March through June. It is rare under the age of 6 months and over 40 years. The incubation period is 14 to 21 days. Transmission of the disease is possible from onset of symptoms to 48 hours after eruption has occurred. The disease produces a life-long immunity. There are conflicting reports regarding the possibility that the administration of gamma globulin late in the incubation period may alter the course of the disease or the immunity produced by it. The clinical picture of the disease is generally well known.

There are no diagnostic tests for rubella, although it has been proposed that significant numbers of Turk and plasma cells in the peripheral blood smear may be significant. There is no general agreement that this test is particularly valuable.

Complications of rubella include polyarthritis, thrombocytopenic purpura, and encephalitis. Polyarthritis is usually mild and self-limited and may resemble the clinical picture of rheumatoid arthritis. Thrombocytopenic purpura is becoming recognized with increasing frequency in recent literature. Encephalitis is fortunately rare, occurring about once in every 6000 cases. Although there are no known permanent sequelae to postrubella encephalitis, one reviewer has calculated the mortality rate to be 20%.

The mode of action of the virus in the rubella syndrome is unknown. The virus may invade various types of differentiating and dividing cells in the fetus, thus producing anomalies by preventing normal development. Once organogenesis is completed and the cells are no longer in active division, susceptibility to the rubella virus diminishes. Another theory suggests that the virus invades only embryonic vascular tissue. By damaging the embryonic blood vessels, nutrition is disturbed during the period of organogenesis.

Congenital Anomalies. 1. Eye Defects. —Approximately 50% of infants with congenital anomalies following maternal rubella will develop some ocular defect. The critical period for the eye is the 4th to 10th week of gestation.

When cataracts result, they are present at birth and are usually subtotal in type and centrally located. They are usually bilateral and the lens and cornea may be smaller than normal. Surgical correction is difficult and frequently unsatisfactory. Because of associated anomalies such as retinopathy, removal of the cataract may not improve vision.

Corneal cloudiness has occasionally been observed. In some cases when the cloudiness has cleared, an underlying cataract or retinopathy may become visible.

Retinopathy is most frequently seen in children who have deaf-mutism without cataracts, or it may be seen in the opposite eye when a cataract is present. Vision may be affected, but there is no progression of the lesions.

The iris in infants with the rubella syndrome may appear atrophic and react weakly and slowly to light. Other ocular defects such as shallow anterior chamber, buphthalmos, nystagmus, and glaucoma are infrequent and are usually associated with one or more of the lesions previously described.

2. Congenital Heart Disease. —The exact incidence is unknown. The most frequent defect is patent ductus arteriosus which occurs in approximately 30% of affected infants. Other conditions include ventricular septal defect, atrial septal defect, and tetralogy of Fallot; and, to a much lesser extent, pulmonary stenosis, aortic stenosis, coarctation of the aorta, transposition of the great vessels, and Eisenmenger's complex.

The exact cause for the increased incidence of congenital heart lesions is not known. It has been suggested that the more frequent involvement of the patent ductus arteriosus is due to the fact that the ductus is open throughout pregnancy and may be damaged at any time, while the critical period for the valves, septa, and great vessels exists only during the first 4 or 5 weeks.

3. Dental Defects. —Incidence of these conditions is also unknown. The lesions which have been reported include hypoplasia of the enamel, pointed incisors, increased incidence of caries, and delay in eruption. The critical period of gestation for the teeth would appear to be between the 6th week and the 9th week.

4. Microcephaly and Mental Retardation. —These conditions—the incidence of which is unknown—occur independently, together, or more

commonly in association with other congenital anomalies. The mechanisms involved in development of central nervous system pathology are poorly understood.

5. Deafness. —The reported incidence of deafness varies from 20 to 70%. The critical period for the ears is between the 1st and 9th week of gestation. Hemorrhage into the middle ear, central neural damage, and lysis of the cells of the basilar membrane of the cochlea have all been reported as possible etiologic mechanisms. Mutism is a frequently associated finding in these children who have severe impairment of hearing and are probably aphasic because of the deafness.

6. Failure to Thrive. —After maternal rubella, infants may be born weighing less than average. Weight may be subnormal even though the child is born at, or near, term. Even though anomalies are present, feeding and growth problems may be more marked than would be expected as a result of the anomaly. Feeding problems and failure to thrive are most often seen in children with patent ductus arteriosus; if this anomaly is not present, the problem is usually less severe.

7. Complications of Pregnancy. —Approximately 10 to 20% of pregnancies complicated by rubella result in stillbirths or spontaneous abortions. The factors causing death in utero are unknown. One theory states that after organogenesis is completed the virus causes the death of the fetus instead of producing anomalies.

Prophylaxis of the Rubella Syndrome. According to current reports in the literature on the incidence of congenital anomalies following maternal rubella, development of the disease during the first trimester of pregnancy carries a risk of approximately one chance in six that the offspring will either be stillborn or have congenital anomalies. Several courses of action are available in an effort to prevent such an occurrence.

1. Inducement of the Disease. —Although this method was formerly considered an effective method of reducing the incidence of congenital anomalies associated with rubella acquired during pregnancy, Schick feels that this would not be a satisfactory answer since the virus may be transmitted to the fetus even if the mother is immune.

2. Passive Immunization. —This form of protection utilizes convalescent rubella serum, gamma globulin, or convalescent phase gamma globulin to produce passive immunity in the individual at the time of exposure to rubella.

Gamma globulin should not be given to a nongravid female because the risk of complications from rubella is slight and the gamma globulin may prevent the individual from obtaining the immunity which follows the natural disease. Despite its limitations and possible ineffectiveness, gamma globulin is the best agent available to produce passive immunity against rubella. Convalescent phase gamma globulin has been demonstrated to give greater protection against rubella than regular immune gamma globulin and convalescent phase plasma. Unfortunately, the agent is not generally available.

There is one problem common to use of all three of these agents: their use may mask the typical picture of rubella and result in rubella without a rash. This may produce a false sense of protection in the patient and her physician, even though the fetus may have been affected by the atypical disease.

3. Therapeutic Abortion. —No specific rules can be set down for use of this measure; each case must be decided on its own merits. Some obstetricians feel that infertility problems are commoner after a therapeutic abortion has been performed.

* * * * * *

Enzymes in Myocardial Infarction

Milton W. Hamolsky MD and Nathan O. Kaplan PhD, Department of Medicine, Harvard Medical School, Boston, Mass. Symposium on Coronary Heart Disease: Measurements of Enzymes in the Diagnosis of Acute Myocardial Infarction. Circulation 23: 102-110, January 1961.

A significant recent advance in diagnosis of heart disease has resulted from development and widespread application of methods to quantitate levels of enzyme activities in the blood or serum of man. The major enzyme systems of present practical value are the transaminases (glutamic-oxalacetic and glutamic-pyruvic transaminase) and lactic dehydrogenase. An increase in the serum level of activity of these enzymes, in the appropriate clinical setting, has proved to be a valuable laboratory indicator of myocardial damage.

Transaminases

Biochemical Background. The term "transaminase" (also called "aminopherase") refers to a group of enzyme systems that catalyze the intermolecular transfer of an amino group (NH_2). Studies have revealed that transamination is probably the most important metabolic mechanism in both formation and deamination of many amino acids in various tissues. There are two separable major transaminating enzymes in heart muscle—glutamic-oxalacetic transaminase (GOT), and glutamic-pyruvic transaminase (GPT). These two systems are widespread in blood and tissues of animals (in man, in descending order of concentration: heart muscle, skeletal muscle, brain, liver, kidney, testis, lung, and spleen).

Methods of Measurement. At the present stage of development, the available methods of enzymology measure rates of reactions; they reflect over-all activity and not the actual concentration of a specific enzyme molecule. This situation necessitates a mental reservation and stresses the completely empirical status of current methods. Furthermore, confusion

has resulted from the early multiplicity of methods with the resultant differences in "normal" and "abnormal" values, necessitating critical evaluation of the specific methods employed in the reporting laboratory.

Clinicopathologic Correlations. Under normal conditions, the abundant transaminases are confined almost exclusively within tissue cells, and only very small amounts are found in the circulation. This situation has permitted ready detection of increased serum values resulting from destruction of tissue and the assumed release of enzyme from the large tissue stores.

SGOT is increased above normal during the first few days after myocardial infarction in man. A significant rise has been detected within 6 to 12 hours of the estimated transmural infarction, generally reaching a peak some 2 to 15 times normal levels in 24 to 48 hours and returning to normal range by the 4th to 7th day. Significant elevations of SGOT, although short-lived, have been found with experimentally induced infarcts of less than 1 gm of myocardium; additionally, serum enzyme levels have not been increased in the presence of significant myocardial ischemia without histologic necrosis.

In man, with obvious limitations of precise definitions, a generally rough correlation of extent of damaged tissue and increase of enzyme activity has been suggested. Actually, the level is of little prognostic value in any given case.

SGPT is frequently normal in the face of elevated SGOT in less extensive infarctions, but may rise in the presence of large infarcts.

Critique. Extensive experience suggests that determination of SGOT (and to a lesser extent SGPT) is an extremely valuable diagnostic tool. Of particular value are rises in enzyme levels in a patient with a suggestive history whose ECG is atypical or obscured by previous myocardial infarction, digitalis administration, bundle-branch block, or the Wolff-Parkinson-White syndrome. A secondary rise in a patient with recent myocardial infarction is an important indicator of extension of the necrotic process. Failure of serum enzyme activity to rise in most cases (usually all but the most severe) of pulmonary embolism or infarction, pericarditis, rheumatic carditis, cardiac arrhythmias, status anginosus, coronary insufficiency, or occlusion without infarction serves as a most useful adjunct in differential diagnosis of chest pain syndromes. An alert must be kept for "false negatives." A major consideration relates to the timing of the specimens for the elevation may occur early, reach a peak transiently within 24 to 48 hours, and return to normal levels by the 2nd or 3rd day. In other instances, elevations may not occur until 36 hours after the onset of symptoms and may persist until the 7th day. Thus, serial determinations are necessary; various sampling schedules have been adopted.

The level of SGOT activity appears to be unaffected by the presence of several other major disease processes, such as heart failure, various infectious diseases, metabolic and endocrine diseases, arthritis, anemias, renal shutdown, venous hypertension, or by administration of digitalis or quinidine.

Activity levels do not correlate with various other factors such as age, sex, race, alterations in leukocyte count, sedimentation rate, body temperature, or urinary volume.

It is pertinent to note other disease processes that may lead to elevated values, although the clinical picture usually suffices to exclude them from serious consideration. Thus, transaminase levels are increased in virtually every type of liver disease; however, differential diagnosis is aided by other liver function tests and by the usually greater elevation of SGPT values in cases of liver-cell injury. Other pathologic states that may be associated with increased enzyme levels are: (1) intracardiac surgery, (2) administration of salicylates, opiates, or coumarin-type anticoagulants, (3) various forms of primary muscular and neuromuscular diseases, (4) acute pancreatitis, (5) extensive central nervous system damage, (6) toxemia of pregnancy, (7) hemolytic crises, (8) crush injuries or burns, and (9) infarction of kidney, spleen, or intestine.

Lactic Dehydrogenase

Biochemical Background and Methodology. Lactic dehydrogenase (LDH) refers to a group of enzymes that catalyze the reversible oxidation-reduction reaction between lactate and pyruvate, involving pari passu the conversion of the reduced and oxidized forms of the pyridine nucleotides (DPNH and DPN).

This reaction is a critically important step in the carbohydrate metabolic cycle and, accordingly, the enzyme is widespread, having been found in virtually every vertebrate and invertebrate species tested.

Recent methodologic advances have culminated in development of a relatively simple, "bedside" method of sufficient accuracy. However, different "normal ranges" have been established by various clinics using modifications of the basic technic.

Clinicopathologic Correlations. The high content of LDH activity in cardiac muscle and relatively low serum values suggested the possible usefulness of study of serum levels in cases of myocardial damage. In characteristic cases, serum LDH activity has been found to be significantly increased above normal at some interval from 12 hours to 10 days after infarction. In general, enzyme activity is increased within the first 12 to 24 hours, rises to a peak in 3 to 4 days, and gradually returns to normal by the 8th to 14th day. Thus, the optimal period of detection is from 2 to 6 days after infarction. Increased levels have occurred even in the absence of definitive ECG changes or consistent alterations in leukocyte count, sedimentation rate, et cetera. Increased values have been found in some cases of severe congestive failure (usually with "congestive cirrhosis" of the liver), bacterial pericarditis, and severe digitalis intoxication; normal values have been found in patients given coumarin anticoagulants or with heart block, prolonged arrhythmias, ventricular aneurysms, or subacute bacterial endocarditis. Elevated

LDH values have been found in acute viral hepatitis, muscular dystrophy, leukemia, and certain cases of metastatic carcinoma.

Critique. Elevation of serum LDH activity is an extremely valuable indication of myocardial damage in appropriate cases. Although the question of sampling time must be considered in the failure to observe an increased LDH in suspected cases, repeated normal values are strong evidence against myocardial necrosis.

Elevated serum values of LDH, as with SGOT, are basically nonspecific. Recent investigation has indicated that there are striking differences in LDH in different tissues. This may result in the possibility of defining more precisely the tissue of origin of the elevated serum levels. The possibility of delineating the specific tissue source affords considerable promise of a major advance in the specificity and diagnostic accuracy of this (and possibly other) enzyme measurements in diseases of man.

Other Enzymes

Changes in other enzyme activities have been investigated in cases of myocardial infarction. To date, none has proved as consistently accurate, sensitive, or helpful as those discussed, but may show some future value.

Malic dehydrogenase (MDH)—catalyzes an important reaction of the citric acid cycle. Measurement is technically less ideal than that of LDH; serum levels are comparably increased in both experimental and clinical myocardial infarction. In general, the time course parallels that of transaminase. It is similarly nonspecific and shows no direct correlation with LDH levels in given cases.

Aldolase—is an enzyme which shows a reaction pattern similar to that for SGOT.

Phosphohexose isomerase—also follows the pattern of transaminase values; elevated values have been found in liver disease, muscle damage, certain tumors, hemolyzed samples, et cetera.

Oxidase—is exhibited in activity with ceruloplasmin, the blue copper-containing serum protein, and a number of substrates. A high degree of correlation has been found among the total plasma copper level, concentration of ceruloplasmin, and serum oxidase activity in normal subjects as well as in myocardial infarction, pregnancy, and chronic infections.

Several recent studies have attempted to correlate multiple enzyme activities on the same samples. However, at the present time it would appear that the mainstays of laboratory diagnosis continue to be SGOT with the advantage of early confirmation, and LDH which is technically easier and permits evaluation over a longer period of time.

* * * * * *

Vascular Compression at the Shoulder Girdle

CAPT Bernard Gardner USAF MC and MAJ Richard H. Hood Jr USAF MC, USAF Hospital Travis, Travis AFB, Calif. Vascular Compression at the Shoulder Girdle: Analysis of Normal Subjects by Means of Radial Pulse Tracings. Ann Surg 153:23-33, January 1961.

Neurovascular compression syndromes in and about the shoulder girdle have been described frequently in the literature, but the pathologic anatomy involved still remains obscure and the relative roles played by the different anatomic components of this area have not been clearly defined in spite of the many theoretical considerations offered. Adding to this confusing picture is presentation of contradictory material and interpretation. Furthermore, the importance of neurologic compression as opposed to vascular compression is a facet where clinical data have led to much disagreement.

The importance of radial pulse diminution with change of posture in these syndromes was recognized as early as 1912. In the study presented, the authors were interested in determining an accurate incidence of radial pulse diminution in various positions of the neck and shoulder girdle in normal individuals. Second, they attempted to ascertain the relationship of sex, age, and body habitus to these findings. Additionally, they hoped that their results might lead the way to a clearer understanding of these syndromes and perhaps indicate the relative importance of some of the anatomic factors involved as well as to serve as a baseline with which symptomatic patients could be compared.

Shoulder girdle syndromes occur more commonly in women, frequently in the third decade, and are rare in children. Body habitus may or may not play a part. However, the impression is gained that the thin, long-necked individual is more prone to develop symptoms.

The symptoms of the syndrome may be related to any of the roots of the brachial plexus, to purely vascular compression, or to a mixture of both. Proof that chronic compression of the subclavian artery in the neck with or without thrombosis can produce symptoms is not available, but the likelihood is difficult to ignore.

Symptoms are more prone to develop at night and may be related to the lying position with accidental hyperabduction of the extremities. They seem to be accentuated by pregnancy, but no satisfactory explanation is presently available.

Certain points may be made concerning the various anatomic components of the neck and shoulder girdle and the role they play in these syndromes:

Cervical Ribs. Neurovascular compression involving the upper extremities is prone to develop in people with cervical ribs. Angulation of the nerves and vessels over a cervical rib may produce symptoms by means

of traction produced by excessive use, or a downward depression, of the extremity. Thrombosis of the subclavian artery has been demonstrated at these points of compression. When a complete evaluation of the patient has shown that cervical rib or ribs are responsible for the symptoms, excision of the rib or ribs is a treatment of choice to prevent serious complications.

First Dorsal Rib. Several reports have indicated a first normal dorsal rib as the agent producing compression by angulation and traction on the neurovascular bundle. Very careful evaluation is necessary before resection of a normal rib should be undertaken as treatment.

Fibrous Band. These may be extensions of partially formed cervical ribs in which case they insert on the first rib and produce traction because of angulation on the neurovascular bundle.

Scalenus Anticus Muscle. This often has been invoked as an etiologic factor by compression of the lower trunks of the brachial plexus against the first rib. There is reasonable doubt as to this muscle producing any effect resulting in symptoms. Recent reports have indicated dissatisfaction with results of scalenus anticus sections for this syndrome, once again indicating that caution should be used before symptoms are attributed to what is usually a normal anatomic condition.

Scalenus Medius Muscle. A percentage of scalenus medius muscles will have a sharp and partially fibrosed anterior border around which the brachial plexus is angulated. This condition is difficult to evaluate; its relative importance depends solely on the accuracy of the exploring surgeon.

Scalenus Minimus Muscle. This muscle occurs in over 50% of normal individuals; it is difficult to understand why this should produce symptoms in a few. It is difficult to visualize the sectioning of this muscle as a treatment for the syndrome.

Clavicle. Compression of the neurovascular bundle by narrowing of the costoclavicular space has been implicated, but serious doubts as to the existence of clavicular compression have been raised by some.

Pectoralis Minor (Sub-acromio-humeral compression). This muscle may play a part in the hyperabduction syndrome which may be a true mechanism producing compression of the vessels and nerves. This may be accentuated by occupations or sleep habits. Treatment appears limited to changing these habits and no surgical correction has been accomplished to date.

Omohyoid. This muscle has been implicated in several specific instances, but accurate data are lacking as to the frequency that this may be a cause of the syndrome.

Brachial Plexus. There have been some detailed data concerning prefixation and postfixation of the brachial plexus. However, certain factors seem to indicate that this is not an important consideration.

Scoliosis of the Thoracic Spine. This condition appears to be a relatively unimportant factor when it is noted that most individuals with severe

scoliosis of the dorsal spine do not have symptoms of upper extremity neurovascular compression.

At the beginning of the investigation, the authors thought that the appearance of a bruit was of significance in the cervical region, especially in the supraclavicular area. As a result of their studies, they now believe that a bruit in this area is of no great diagnostic aid.

On analysis of their data, the authors consider that two factors seem to be of importance in producing vascular compression at the shoulder girdle: lifting the arm above the head (the so-called hyperabduction position), and bracing the shoulders (moving them backward and downward). The factor that is common to both is the tensing of the pectoralis minor. The authors' opinion is that frequent use of the extremity under these abnormal positions can lead to symptoms.

* * * * * *

Juxtaglomerular Cells and Hypertension

Claire Turgeon MD and Sheldon Sommers MD, Department of Pathology, Massachusetts Memorial Hospitals, Boston, Mass. Juxtaglomerular Cell Counts and Human Hypertension. Amer J Path 38: 227-242, February 1961.

The juxtaglomerular body of the kidney was classically described by Goormaghtigh. It consists of 8 to 15 epithelioid cells located near the glomerular hilus, apparently in continuity with the muscle cells of the afferent arteriole, and in close proximity to the macula densa. Together, the juxtaglomerular body and macula densa compose the juxtaglomerular apparatus. The function of these morphologic entities is not known with certainty, although the juxtaglomerular cells have been thought by many to have an endocrine action. In recent years, attempts have been made to correlate the granularity of the renal juxtaglomerular cells with hypertension, blood electrolytes, and the effects of mineralo-corticoid hormones. An attempt was made by the authors to correlate the morphologic features in surgical specimens with clinical observations in patients with hypertension.

The most distinctive alteration observed in the juxtaglomerular body in surgical tissues was the significant cellular hyperplasia in cases of hypertension that followed some occlusive process in one renal artery and represented typical examples of primary renal hypertension. This was most striking in younger patients, presumably without other serious vascular lesions, with only slight to moderate renal atrophy, and with hypertension of short duration. These characteristics are regarded clinically as good indications for unilateral nephrectomy which is often followed by a cure of the hypertension. The hyperplastic aspect of the juxtaglomerular body in a renal biopsy may have an important diagnostic and prognostic value in such cases.

Specimens from the contralateral kidney, when available, showed that both the total counts and the types of cells believed to be functionally more active were significantly decreased in the unaffected kidney.

These observations favor the possibility of an endocrine function in the juxtaglomerular cells. They could secrete some hypertensive factor like renin, either following ischemia or, more probably, in response to some hemodynamic change within the kidney. Hyperplasia ordinarily indicates increased function in many other secretory cells of the body.

Clear watery cells presumably reflected an excess in both hormone production and secretion. The increase in the number of cells was, in general, more significant and reliable than modifications in their granularity. Individual variations in granularity were wide, but the deviations appeared significant only in the "Goldblatt type" cases and in essential hypertension with grade III arteriolar sclerosis.

The nature of the granules is unknown; they may represent stored vasopressor substance or a precursor of the active proteolytic enzyme, renin. A close correlation between renin extracted from the kidney and the number of granules in the juxtaglomerular cells has been shown in animals. Analogous results in the human subjects studied by the authors were not obtained.

Results of the authors' study are highly suggestive of the presence of a vasopressor substance like renin or a precursor in the juxtaglomerular cells. Proteolytic enzymes seldom exist as such intracellularly, and some autolysis is necessary before renin can be extracted biochemically from renal tissue.

The problem is still unsolved in cases of pyelonephritis with hypertension. No definite juxtaglomerular alteration was found. Patients with unilateral pyelonephritis, in general, had a moderate hypertension for several years; their average age was 53, plus or minus 15 years, and they were not cured of their hypertension by a nephrectomy—with one exception.

In pyelonephritis with hypertension, the arcuate and interlobular arteries have thicker walls and a more reduced lumen than in pyelonephritis without hypertension. There may be a prolonged low-grade internal ischemia or hemodynamic modification in these cases with hypertension as a final result. Further investigation would be desirable to prove this hypothesis.

In the examples of essential hypertension investigated, a significant increase in the total number of juxtaglomerular cells was noted. Similar features were observed in the kidney tissues from patients with pheochromocytoma. In the latter groups, the change was very likely secondary to intermittent secretion of catecholamines, spasm, and anatomic alterations in the renal vasculature. This similarity does not exclude a primary renal vasopressor factor in essential hypertension. On the other hand, secondary changes both in renal vessels and juxtaglomerular cells could contribute renal factors with the property of sustaining early functional hypertension and making it irreversible.

If large clear cells are presumed to be hyperactive and small cells nonfunctioning, the reduced number of clear cells in "malignant" hypertension appears paradoxical. It is possible that a blood pressure high enough to cause arteriolar necrosis may also inhibit the juxtaglomerular secretion by a mechanism opposite to that found in "Goldblatt type" kidneys. The increase of hypertensin once reported in such cases would remain unexplained since renin is thought to be necessary to transform hypertensinogen into hypertensin.

The findings in glomerulonephritis are in agreement with the original description by Goormaghtigh, as were those in the "Goldblatt type" kidneys. There was hyperplasia in early stages, regardless of the level of blood pressure, and atrophy in the extensively scarred kidneys. It may be necessary to invoke a factor such as modification in the enzymatic system for degradation of pressor amines, to explain the persistent hypertension in patients with severely damaged kidneys and atrophic juxtaglomerular bodies as were found in chronic glomerulonephritis, advanced chronic pylonephritis, and protracted "Goldblatt type" kidney lesions.

* * * * * *

Live Polio Vaccine

Saul Krugman MD, Marvin S. Eiger MD, et al, Department of Pediatrics, New York University School of Medicine, 550 First Ave., New York City. Immunization with Live Attenuated Poliovirus Vaccine. Amer J Dis Child 101:23-29, January 1961.

Live attenuated poliovirus vaccine was administered to 400 newborn infants during a 3-month period in a study designed to answer the following questions:

1. Would ingestion of larger doses permit enough of the vaccine viruses regularly to pass the "acid barrier" of the stomach of newborn infants and result in regular multiplication in the intestinal tract? (Poliovirus may be destroyed below a pH of 2.5 and the gastric contents of newborns often have a pH of about 1.5.)
2. Would it be possible to bypass the potential handicap of high gastric acidity by swabbing the vaccine directly on the posterior pharyngeal wall?
3. Would all three types multiply following administration of a mixture of large doses of Types 1, 2, and 3 poliovirus either by mouth or by throat as determined by virus excretion and antibody formation?

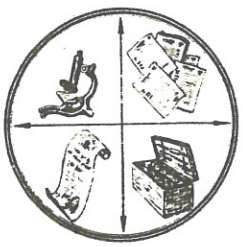
Demonstration of virus multiplication in the intestinal tracts of 80 to 90% of newborn infants fed the live vaccine was a significant finding. It would appear that the factor of high gastric acidity in the newborn infant does not present a serious handicap in oral immunization when larger doses of vaccine are used.

Presence of high levels of maternally transmitted antibody had no effect on the extent of multiplication of virus in the intestinal tract.

Evidence of an active antibody response was more apt to occur in infants excreting virus than in those with negative tests for virus in single stool specimens. Equally noteworthy was the fact that in spite of active multiplication of virus in the intestinal tract, 58% of the infants had no evidence of an active antibody response on the basis of the criteria that had to be adopted. Although there was no correlation between the titer of antibody in the cord blood and the extent of virus multiplication in the intestinal tract, the reverse was true for the relationship between antibody titer at birth and the evidence for active antibody response at 3 months—the lower the titer the higher the percentage of responses.

Absence of evidence of an antibody response in an infant with a high titer of antibody in the cord blood does not necessarily mean lack of active antibody production.

* * * * * *



MISCELLANY

Rear Admiral Allan S. Chrisman to be Deputy to the Surgeon General

Rear Admiral Edward C. Kenney has selected Rear Admiral Allan S. Chrisman to serve as Deputy Surgeon General and Assistant Chief of the Bureau of Medicine and Surgery during his four-year appointment as Surgeon General of the Navy. Admiral Chrisman, who is Commanding Officer of the U. S. Naval Hospital, San Diego, Calif., and District Medical Officer, Eleventh Naval District, has received orders from the Bureau of Personnel and is expected to report to the Bureau about 10 April 1961 to assume his duties.

This will be the second assignment in the Bureau for Admiral Chrisman, his first tour being from 1952 to 1956 when he was the Head of Personnel. Following

Bureau duty, Admiral Chrisman was sent to the hospital at San Diego as Commanding Officer and in 1958 he was assigned additional duty as District Medical Officer in that district. Relieving him in San Diego will be Rear Admiral Harold J. Cokely who has been Commanding Officer of the St. Albans Hospital.

Admiral Chrisman was born in Greensboro, N. C., and received his early education in that State—public schools in Charlotte, and Bachelor of Science degree from the University of North Carolina in 1926. He was commissioned in the Medical Corps of the Navy on 3 June 1930, after receiving the degree of Doctor of Medicine from Harvard Medical School. After internship at the Naval Hospital, Philadelphia, he was a student at the Submarine School, New London, Conn., and subsequently served as Medical Officer aboard the USS S-4 from December 1931 to April 1933. During that time he made a tour of all Submarine Bases in connection with submarine escape training. He next served for nine months as Medical Officer of the USS BEAVER, tender for Submarine Squadron 4, based at Pearl Harbor.

During the next six years Dr. Chrisman served at the Naval Hospital, San Diego; was Medical Officer at the Submarine Base, New London, Conn.; and Medical Officer aboard the USS RANGER on Neutrality Patrol in the Atlantic.

In 1940, Dr. Chrisman received graduate instruction in Radiology at the Naval Hospital, Washington, D. C., following which he served in the X-ray Departments of the Hospitals at Parris Island, S. C., and Bethesda, Md. In September 1942, he joined the USS PINKNEY and, as her Medical Officer, saw action in the South Pacific Area. In August 1943, he reported as Base Medical Officer at the Advanced Naval Base, Tulagi, Solomon Islands, and had charge of the Tulagi-Florida Medical Facilities.

In January 1944, Dr. Chrisman returned to New London, Conn., to be Assistant Officer in Charge of the Medical Research Laboratory. In connection with this duty he was awarded a Letter of Commendation, with Ribbon, from the Commander-in-Chief, U. S. Atlantic Fleet, "for meritorious service." From January 1945 to July 1950 he served consecutively at Naval Hospitals at Bainbridge, Md. (Executive Officer), Newport, R. I. (X-ray), Aiea Heights, Hawaii (X-ray), and Camp Lejeune, N. C. (Executive Officer). Following a year as a student at the Naval War College, Newport, R. I., he served as Force Medical Officer to Commander Submarines, Atlantic until he reported to the Bureau in 1952.

Admiral Chrisman is a member of Phi Kappa Sigma, Phi Chi, and Phi Betta Kappa fraternities. He is married to the former Eleanore Krekeler of Montclair, N. J.; they have three children, Caroline, Jane, and Allan. Mrs. Chrisman and the children will join Admiral Chrisman in Washington at the close of the present school year.

* * * * * *

Admiral Hogan Honored in Retirement Ceremony

On 28 February 1961 at the National Naval Medical Center, Bethesda, Md., Rear Admiral Bartholomew W. Hogan, Medical Corps, U.S. Navy, was honored by a colorful military ceremony marking his retirement from active duty after nearly thirty-six years of service. Admiral Hogan, recently completing a six-year term as Surgeon General of the Navy, was additionally honored by being awarded the Distinguished Service Medal in recognition of his leadership and accomplishments during that period.

Following the reading by Admiral Hogan of orders which retired him from active duty, a message from Admiral Arleigh Burke, Chief of Naval Operations, was read and appropriate remarks were made by the Chief of Naval Personnel, Vice Admiral William R. Smedberg. Admiral Smedberg then presented the Secretary of the Navy, the Honorable John B. Connally. After reviewing Admiral Hogan's distinguished career, Secretary Connally presented him with the Distinguished Service Medal.

During remarks just before reading his final orders, Admiral Hogan said: "Nothing begins that does not end. In every drama there is a final curtain. In every career there is a peak; and when that point of height is reached . . . everything falls into proper perspective."

"On June 6, 1925, I had the honor—and it was an honor," he continued, "to be appointed a Lieutenant (jg) in the Medical Corps of the U.S. Navy. The gold on my sleeves was bright. . . . It would only be human to admit that I was proud to be a doctor—and proud to be an individual unit as an officer of the U.S. Navy. To the men whom I have met since that June day of 1925, I have been bound in a close community of effort. . . . No one can be part of the Navy and be immune to the traditions of the past, the vitality of the present, the brightness of the future. No one can be of the Navy and not grow mentally, physically, and spiritually."

"To the Navy I owe a debt of thanks," he stated. "I am glad—I am proud—I am content. I am satisfied that I have spent—and given—36 years of my life to such a cause. To the men of the past and the men of the present whom I have known, enlisted men, officers, civilians, their wives and families, I boast that I count you as my friends. Never for one moment during my career have I forgotten that the highest could not exist but for the lowest. I know that the gold on my sleeves this afternoon shines with brighter pride than it did 36 years ago because of what you and I have done together."

Admitting to some recently experienced regret that his active duty was soon to be terminated, Admiral Hogan said: "However, after a period of contemplation, these regrets evaporated and were replaced by a great optimism based on the following knowledge. First, the gentleman and officer who was selected to relieve me as Surgeon General, Dr. Kenney, is the best qualified person to have been selected for this position during the period of my years in the Navy. He is outstandingly fitted professionally and administratively,

a great leader with a brilliant mind. Second, the various components of the Medical Department . . . a total of approximately 41,000, comprise a group of professionally competent and dedicated people who have few equals in the field of medicine." A third reason for optimism, Admiral Hogan considered to be the increasingly higher level of education and training seen in personnel entering the Medical Department in recent years, which he believes is laying a solid and firm foundation for a greater future.

The ceremony which included the stirring thirteen-gun salute, ruffles and flourishes and Admiral's March by the Navy Band, and inspection by Admiral Hogan of the Honor Guard composed of sailors and Marines, concluded with orders to haul down the Admiral's personal flag. It was then folded and presented to Admiral Hogan by his son, LT Bartholomew T.Hogan MC USN who is serving an internship at the U.S. Naval Hospital, Bethesda.

* * * * * *

BUMED INSTRUCTION 6710.43A 9 February 1961

Subj: Parenteral solutions; cautions regarding

Experience has repeatedly demonstrated the continuing necessity for close and detailed examination of all parenteral solutions and their containers — at regular intervals while in shelf stock and prior to their use. This careful examination is mandatory because of the possibility of: (a) deterioration from prolonged storage, (b) accidental contamination occurring in the original packaging, and (c) defects which may develop in the container and/or its seals. Administration of contaminated or defective parenteral solutions is a professional accident which might well be fatal. This directive reemphasizes the urgent necessity for carefully examining and meticulously scrutinizing all parenteral solutions before they are administered to patients, and describes specific examination procedures.

* * * * * *

BUMED INSTRUCTION 6710.46 23 February 1961

Subj: Caution concerning use of chloramphenicol

Despite recurring reports of bone marrow depression following administration of chloramphenicol, which frequently results in fatalities, the antibiotic continues to be used without sufficient indication (Medical News Letter, p. 21, 17 February 1961). This directive cautions all Medical Department personnel against indiscriminate use of chloramphenicol and refers to BUMEDINST 6710.2 which specifically directs that the antibiotic should not be administered without a specific order of a Medical or Dental officer.

BUMED NOTICE 6120 9 February 1961

Subj: Physical examinations; conducting and reporting results of

Unnecessary correspondence concerning inaccuracies or required additional medical information could readily be prevented by closer adherence to prescribed methods of performing and reporting physical examinations. This directive encourages careful review of the requirements in the Manual of the Medical Department and current directives concerning physical standards and examining procedures and proper reporting of results by medical examiners. It specifically lists frequently recurring errors and discrepancies.

* * * * * *

From the Note Book

ADM Hogan APA Director. Following retirement from active duty, Rear Admiral Bartholomew W. Hogan, formerly Surgeon General of the Navy, assumed his duties as Assistant Director of the American Psychiatric Association on 1 March 1961, with offices at 1700 18th Street, N. W., Washington 9, D. C.

USNH Phila Staff in Teaching Role. Affiliation with local medical schools has been announced recently for two staff members of U. S. Naval Hospital, Philadelphia: CAPT Herschel E. Richardson MC USN has been appointed as Assistant Professor of Medicine in the Department of Medicine, Hahnemann Medical College; and LT Robert C. Brod MC USN has been appointed as an Assistant in Medicine at the Jefferson Medical College and Medical Center.

USNH Brooklyn Reunion. The annual reunion dinner of World War II Alumni of the U. S. Naval Hospital, Brooklyn, will be held at the Commissioned Officers Mess (Officers Club) of the U. S. Naval Shipyard, Flushing Ave., Brooklyn 1, N. Y., Thursday, 6:00 p. m., 20 April 1961. Inasmuch as membership in the Alumni Association has been widened to include all Medical officers who served at medical facilities in the Third Naval District, all such officers are invited to attend.

Nuclear Medicine Course at NNMC. Seven Naval Medical officers, four civilians, and one Chilean Navy officer have begun a 12-week course in Radioisotope Technics and Nuclear Medicine at the National Naval Medical Center, Bethesda, Md. (TIO, BuMed)

Indoctrination for Carrier MOs. Plans have been made to establish a 2-week indoctrination period at Norfolk, Va., for new Medical officers who are assigned to Atlantic Fleet aircraft carriers. The indoctrination will cover organization, administration, sanitation requirements, medical intelligence, and other phases of medical department operation peculiar to a carrier.

NNMC Receives Fire Prevention Award. The National Naval Medical Center, Bethesda, Md., has recently been awarded Honorable Mention in the Military Division of the National Fire Protection Association's 1960 fire prevention contest. This was the second time that the Medical Center has received an award in the contest and was the only naval medical activity cited in the 1960 contest. Entry was in the Military Division under medium activities having 1500 to 3500 personnel, with 108 activities being in the competition. (PIO, NNMC)

Influenza and Hepatitis Notes. No reports have been received to date of unusual activity of influenza within the continental U.S. Deaths due to pneumonia and influenza in 108 U.S. cities have remained within the expected limits of normal for the season. The epidemic of A_2 influenza in Great Britain is reported to be showing a significant decline—972 deaths for the week ending 18 February, compared to 1393 deaths for the preceding week. Hepatitis continues to show increasing incidence; 2003 cases were reported for the week ending 25 February, the highest number for a single week since hepatitis became a nationally reportable disease (1952). Increases are occurring in all States and geographical divisions. Cases during 1961 are 128% above a comparable 8-week period last year. (Morbidity and Mortality, PHS DHEW, March 3, 1961)

Hepatitis in Recipients of Screened Blood or Aged Plasma. Because it was suggested that screening blood donors by means of chemical testing at the time of donation might reduce the number of carriers of hepatitis, the author prepared a study of the problem. His conclusions showed that determinations of thymol turbidity, thymol flocculation, and total bilirubin of serum of donors seemed to be of no value in detecting carriers of hepatitis viruses. Furthermore, storage of liquid plasma at room temperatures did not seem to be sufficient to eliminate the occurrence of hepatitis. (N. Maxwell, Amer J Clin Path, December 1960)

Role of Kidney in Production of Erythropoietic Factor. The author's data reveal that bilateral nephrectomy of the anemic dog causes a rapid disappearance of measurable amounts of plasma erythropoietic factor. These experiments add support to the hypothesis that the kidney produces the factor; or, if the kidney is not the source of erythropoietin it may be the site of destruction of an erythropoietin inhibitor. (Jean-Pierre Naets, Blood, December 1960)

DENTAL SECTION

Malpractice Suits Against Dental Officers

The question of malpractice suits against Navy Dental officers arises periodically. The subject is covered in the Manual of the Medical Department, articles 3-29 and 6-36. Some salient facts not covered in the manual are:

1. The dental officer may be sued alone or in conjunction with the Federal Government.
2. Malpractice suits against an officer may include his commanding officer (U. S. Naval Dental Clinic), senior dental officer, or chief of dental service even though they have no part in the treatment.
3. The Federal Tort Claims Act does not constitute a protective umbrella for the Navy Dental officer.
4. The Federal Government does not, of necessity, have to provide council.

If a Dental officer is informed of any civil litigation or legal proceeding being brought against him wherein the United States is, in effect, the defendant, he should immediately advise his commanding officer so that a report can be made, as directed in the 1955 Naval Supplement to the Manual for Courts Martial United States 1951. A copy of this report should be sent to the Bureau of Medicine and Surgery.

The question often arises as to what protection is afforded the service doctor against malpractice or professional liability suits. According to Coward, the service doctor is less likely to be sued in his individual capacity than is his civilian counterpart. This is due to the protection given him by the Federal Tort Claims Act of 1946 and other Federal laws. If a service doctor is sued in a state civil court for acts performed within the scope of his duty, he may have the case removed to a Federal court for trial. He may also arrange to be defended by a United States attorney. Because of this protection, the rates for liability insurance are considerably lower for doctors in the Federal service than for doctors in civilian practice. This is not meant to infer that the Government recommends commercial professional liability insurance for its doctors. The decision as to whether he should purchase such insurance is left up to the individual doctor.

No physician or dentist should be reluctant to prescribe treatment in an effort to protect himself against malpractice claims; he should,

instead, make it a practice to utilize the following safeguards for minimizing possibilities of legal action against him:

1. Avoid careless remarks about treatment given by another doctor.
2. Keep complete and accurate records.
3. Make a thorough examination of the patient, including all necessary clinical and laboratory tests and roentgenograms.
4. Establish and maintain rapport with the patient.
5. Not use unproven drugs or treatment methods.
6. Not guarantee results or fixed degrees of improvement.
7. Explain all risks to the patient.
8. Obtain written consent for proposed procedures when it is appropriate to do so.
9. Use discretion when prescribing treatment or drugs by telephone.
10. Not take photographs of patients without their written consent.

The Armed Forces Medical Journal, February 1958 (page 224), contains an excellent discussion of the subject.

* * * * * *

Anxiety, Apprehension and Fear at the Dental Office

Theodor Anderes, Flawil, Canton St. Gallen, Switzerland. Schweiz Mschr Zahnhk 70: 424-427, May 1960.

Although pain caused by dental procedures is experienced readily, the objective degree of the pain sensation is difficult to measure or even to define accurately.

Most modern dental procedures, however, are performed with little or no physical pain. After completion of dental treatment, many previously apprehensive patients will admit that they did not experience the discomfort, distress, or agony which they had anticipated. But, prior to and during the procedure their anxiety, apprehension, and fear still had persisted.

The dental patient's psyche, therefore, cannot be ignored by the modern dentist who intends to render a complete health service by treating the mind as well as the mouth. With the advent of tranquilizing agents it became imperative that the applicability of these drugs to dental practice should be investigated.

The following properties are prerequisites for any drug to be used at the dental office to induce emotional calm in obviously apprehensive patients: (1) minimal toxicity, (2) rapid onset of action, (3) long-lasting effect, (4) low incidence of side effects and aftereffects, (5) minimal habituation (drug addiction), (6) minimal acquired tolerance, (7) effectiveness and suitability for outpatient treatment of ambulatory patients, (8) inability to dull the senses, decrease the perception, and interfere with

mental acuity, (9) availability in forms suitable for easy administration, and (10) attainability at reasonable cost.

Hydroxyzine hydrochloride (Atarax) has been investigated by several researchers in Switzerland as well as in European and American countries and has been found to be an effective tranquilizing agent for use in dental practice. Its toxicity is extremely low; during the tests the drug was administered in doses exceeding 300 mg daily for several months without untoward effects.

In the present study, hydroxyzine hydrochloride was used prior to 1128 dental procedures performed on 305 patients. It was administered in tablet or dragée form (25 mg daily) and provided adequate sedation in extremely apprehensive patients without causing reduction in the leukocyte count. The degree of sedative effects produced by this drug was significantly higher than that of placebos administered in double-blind tests. Duration of the tranquilizing effect varied between one and two hours, thereby providing ample time to complete most dental procedures. Other than a slightly decreased salivation and a mild dryness of the mouth, no side effects were observed.

Hydroxyzine hydrochloride proved to be a valuable adjunct to the customary anesthesia in dental practice, especially suited for pretreatment administration in hypersensitive patients. (Dental Abstracts 6:32, January 1961)

* * * * * *

Occupational Dermatitis in Dentists

D. A. Wallace and J. P. Weinmann, College of Dentistry, University of Illinois, Chicago, Ill. Internat D J 10:75-88, March 1960.

Occupational dermatitis among dentists is almost always of the contact type. Available surveys indicate that about 3% of all dentists suffer at one time or another from occupational allergic eczematous dermatitis, and that the condition threatens to be disabling to somewhat less than 1% of all dentists.

The history is of the greatest importance in diagnosis. Skin eruptions caused by allergic reactions to drugs can mimic the appearance of any dermatosis. The location and pattern of the lesions often are informative. Of various skin tests, the patch test is the one most highly recommended.

The likelihood of dermatitis increases with frequency and intensity of exposure to irritants. A weakened or diseased skin is more susceptible than a normal skin. Avoidance of the responsible substance usually will be followed by complete remission. Self-treatment is hazardous because many drugs used to relieve pruritis and other dematologic symptoms contain anesthetics and other sensitizers which may aggravate the condition.

Contact dermatitis may be caused by primary irritants or by sensitizers; usually, it is caused by a combination of factors. The most common causes are contact with local anesthetics and instrument, or pulp canal, disinfectants. Excessive exposure to water and hand cleansers may predispose the skin to contact dermatitis. Healing usually occurs soon after exposure to the causative substance is stopped.

In most instances the prognosis is favorable, but once hypersensitivity is established it is likely to persist. The universal remedy is to avoid contact with known allergens and to minimize exposure to cleansers and irritants.

The following specific suggestions to dentists are offered:

1. Wash the hands only when necessary, and only as vigorously as necessary.
2. When possible, use heat rather than chemical methods to disinfect instruments.
3. Handle drugs and dental materials with instruments, to avoid their contacting the skin.
4. When air is expelled from the hypodermic syringe, avoid getting a spray on the face. Do not let the anesthetic solution expelled from the needle come in contact with the skin.
5. Have the patient rinse his mouth with water immediately after injection of a local anesthetic. The dentist at this time may wish to rinse his hands.
6. If necessary, use rubber gloves or finger cots when injecting an anesthetic. Have the rubber items cleaned and dried after each use.
7. Take the best possible care of the hands. If creams or lotions seem desirable, consult a dermatologist and use only those items which he recommends.

(Dental Abstracts 6:47, January 1961)

* * * * * *

Scrap Dental Metals

In January and July of each year, or upon decommissioning or disestablishment, the commanding officer or officer in charge of a naval dental activity, the Dental officer of a ship or station, and the commanding officer of a U. S. Naval Hospital or U. S. Naval Dispensary shall forward, for disposal, all scrap dental metals to the appropriate disposal site in accordance with instructions contained in Chapter 6-166, Manual of the Medical Department.

A study was conducted to determine the value of this scrap metal returned to the supply system. Representative total savings over a 2-year period—$30,667.23 and $46,000.00, respectively, at Supply Center,

Oakland and Bayonne---indicate the importance of this program. Every effort must be expended to insure maximum effective use of dental metals and timely return of all scrap.

* * * * * *

Personnel and Professional Notes

Dr. Peterson Lectures at NDS. Shailer Peterson BA MA PhD, Chicago, Ill., Secretary of the Council on Dental Education, American Dental Association, recently presented a lecture---A Balanced Educational Program for the Professional Man---at the Naval Dental School. Dr. Peterson, speaking to Dental officers of the Armed Forces, civilian dentists, and other scientific personnel of the Washington, D. C., area as part of the special lecture series of the School, discussed the need for postgraduate and continuous training courses for dentists. He stressed the need for dental schools to redouble their efforts to serve their students and alumni in helping them to keep up with the times and equipping them to meet the challenges of a complicated world.

Dental Officers at Chicago Midwinter Meeting. At the recent 86th Midwinter Meeting of the Chicago Dental Society, several Dental officers of the Naval Administrative Command, U. S. Naval Training Center, Great Lakes, Ill., participated. Table clinics were given by: CAPT R. H. Loving---Periodontal Problems Resulting from Faulty Tooth Position; CDR M. L. Hermsmeyer---Gold Cast Directly to Porcelain Facings; CDR M. A. Mazzarella and LT W. R. Shiller---Dental Research at Great Lakes; LT R. N. Draheim and LT R. T. Rydstrom---Condensation of Mat and Cohesive Gold; LT E. E. Houk and LT C. Passalino---Cavity Preparation for Class V Gold Foil; and LT B. J. Grothaus and LT J. B. Schram---The Rubber Dam for Class V Gold Foil. CAPT R. B. Wolcott presented two limited attendance clinics---Successful and Unsuccessful Amalgam Practices.

CAPT Gargiulo Presents Paper. CAPT Edward A. H. Gargiulo DC USN, Chief of Dental Service, U. S. Naval Hospital, Philadelphia, and Diplomate of the American Board of Oral Surgery, recently participated in the In-service Training Program of the Division of Dental Health of the Philadelphia Department of Public Health, discussing Local Anesthetics - Technic and Handling.

CDR Rovelstad Presents Paper. CDR Gordon H. Rovelstad DC USN, U. S. Naval Dental School, NNMC, Bethesda, Md., recently participated in the meeting of the Lincoln District Dental Society at Lincoln, Neb., discussing Responsibility of the Dentist in the Prevention of Dental Caries.

CDR Rovelstad recently was a member of the examining board in the annual examination of the American Board of Pedodontics held at the New York University Dental School, New York City.

* * * * * *

RESERVE SECTION

Vacation Training for Ensigns 1915 USNR

Ensigns 1915 USNR who have had no training duty in fiscal year 1961 and have a vacation period prior to 1 July 1961 are urged to request clinical clerkship training at a naval teaching hospital. All naval districts have been allocated additional quotas for this training. Clerkships available are of 60 days duration or less, but in no instance less than 14 days. Because quotas are limited, interested eligible Ensigns should request this duty from their naval district commandant as soon as possible.

* * * * * *

Are You Eligible For Certain Tax Deductions?

It is now the time to figure your 1960 income tax. Before you "ante up" to Uncle Sam, however, it will pay you to look over some deductions you may have "earned" through participation in the Naval Reserve program. If you are on inactive duty, you may deduct transportation costs involved in attending drills. You may also be allowed to deduct amounts spent for purchase and maintenance of your Navy uniforms. Here is the latest information on income tax deductions for Naval Reservists:

Uniform Costs. If your uniform expenses were not covered by nontaxable allowances or uniform gratuities, you may deduct the amounts spent for purchase and maintenance of uniforms for Federal income tax purposes.

An Internal Revenue Service ruling states that the deduction is allowed as an "ordinary and necessary business expense" when uniforms are required and allowed to be worn only when on active duty for training for temporary periods, and when attending service school courses and training assemblies (drills).

If you are on inactive duty you may deduct not only the cost of uniforms but the maintenance of these uniforms. If, however, you receive a

uniform gratuity, your expenses are deductible only to the extent that they exceed your uniform gratuity in that particular year.

For example: You may deduct 1960 uniform expenses---purchase price and maintenance costs---when you file your 1960 Federal income tax return. If you received a uniform gratuity in 1960 of, say, $100 and the cost and maintenance of your uniforms amounted to $150, you may deduct $50 on your tax return. If you received no uniform gratuity in 1960, you may deduct the entire sum---in this instance $150. A uniform gratuity received in 1960 need not be considered except with respect to expenses incurred in 1960.

Reservists serving on full-time active duty may deduct only the cost of all items of insignia of rank and corps.

Travel and Transportation Expenses. Travel and transportation allowances paid by the Navy Department while you are in a mileage or per diem status are considered to be an accounting to your employer. If you broke even—or if you do not choose to deduct excess expenses—you may simply answer "yes" to the question relating to travel on page 1, Form 1040 or 1040W, or check item 8, page 1, Form 1040A, and forget the matter.

However, if allowances exceeded expenses, you should answer "yes" to the questions on page 1 of Form 1040 or 1040W and enter the excess, labeled "excess reimbursements," as "wages."

If you claim excess expenses---or if no allowances were authorized---all allowances, reimbursements, and expenses must be listed. The excess expenses are computed on IRS Form 2106 and deducted from your Navy pay, if any, before entering the net wages or expenses as "wages" on page 1 of Form 1040 or 1040W.

"Travel expenses" include meals and lodging of Reservists who, under competent orders and with or without compensation, are required to remain away from their principal place of business overnight in the performance of authorized drills and training duty.

Reservists required to work and drill on the same day at each of two different locations within the same city or general area may deduct one-way "transportation expenses" in going from one place of business to another. When they return home before drills, one-way expenses from home to place of drill, not to exceed expenses from place of work to place of drill, may be deducted. However, round trip transportation expenses are deductible when the duty area is situated beyond the city or general area which constitutes the principal place of business, provided free transportation between these locations is not furnished by the Navy.

There is no authority in either the Internal Revenue Code or regulations for a flat rate-per-mile deduction for transportation expenses when you travel in your own automobile.

Expenses of an automobile would ordinarily include such items as gasoline, oil, minor repairs, depreciation, and the like. If you keep a

record of all automobile expenses for the year, you can easily determine the amount of deduction for your drill trips. One way to do this is to take the ratio of the total mileage of your drill trips to the total mileage for the year and apply that percentage to your total expenses for the year.

The Internal Revenue Service has accepted a reasonable rate-per-mile in lieu of actual automobile costs under certain circumstances—up to seven or eight cents per mile. This is a "rule of thumb" practice, however, and has no basis in law or regulation.

Additional information on income tax deductions may be found in the Federal Income Tax Information for Service Personnel pamphlet prepared annually by the Judge Advocate General. Copies of this publication should be available at your Naval Reserve Training Center or the nearest naval activity.

If you were released to inactive duty and have since lost or misplaced your Withholding Tax Statement (Form W-2), you may request a copy of this form from the Commanding Officer, U.S. Navy Finance Center, Cleveland 14, Ohio. (The Naval Reservist, February 1961)

* * * * * *

OCCUPATIONAL MEDICINE

Primary Irritant Nature of Cement

Portland cement and similar cements used in the building trade for reinforced concrete, mortar, plaster, et cetera, consist of lime, clay, and silica in varying proportions. "Cement itch," or dermatitis, has been known for years and is common among workers handling such products. Recently, much has been written about the part chromium salts play in the cause of such dermatoses. European writers, especially, believe that cement dermatoses are due in large measure to sensitization to water soluble chromate compounds present in the cement. There is no adequate proof, however, that sensitization to chromate or dichromate is the major factor in cement dermatitis. Nowhere has the author been able to find any note of primary ulcers of the skin due to the action of cement. In the present paper the author reports on two such cases. (G.E. Morris, Arch Environ Health, October 1960; abstracted in Industr Hyg Dig, January 1961)

The Toxicology of Carbon Tetrachloride

Charles E. Lewis MD Sc D, University of Kansas School of Medicine, Kansas City, Kans. J Occup Med, 3: 82-85, February 1961.

Production of carbon tetrachloride in the United States was 374,000,000 pounds in 1959; probably more than 50% has been or will be used in the manufacture of other chemicals, such as dichlorodifluoromethane (Freon 12). Much of the remainder will be used in fire extinguishers, fumigation of grain, manufacture of rubber, and as a degreasing agent or solvent. A large quantity of carbon tetrachloride will find its way into the hands of the unwary public. It will enter their homes as a constituent of several popular cleaning agents, in insecticide sprays, or upon the recommendation of a friend and by direct purchase of "carbon-tet" from drugstores, hardware stores, and establishments selling and servicing fire extinguisher equipment.

A case of carbon tetrachloride poisoning serves to illustrate the relative importance and interaction of the three basic factors in the pathogenesis of disease---the agent, the host, and the environment. The agent, carbon tetrachloride, must be available in sufficient quantity for the host---or victim---to absorb a toxic amount. In cases of intoxication due to inhalation, the environment must permit the accumulation of toxic concentrations of carbon tetrachloride in the ambient air. This usually means a small unventilated room. Certain factors in the host predispose to development of pathology following exposure to carbon tetrachloride. Obesity, excessive intake of alcohol, presence of liver disease serves to increase the susceptibility of the host. Perhaps the most important contribution of the host is the selection and misuse of a potentially dangerous material, usually because of ignorance of its toxic properties.

Poisoning in Man

Carbon tetrachloride poisoning may be acute or chronic. It can be due to ingestion of the liquid or inhalation of its vapors. Acute intoxication results from inhalation of vapors produced while using the material in a small unventilated area, intentional ingestion with suicidal motives, or accidental ingestion (most common to small children and alcoholics). Chronic intoxication is usually due to industrial exposures.

Acute Intoxication Due to Inhalation. Exposure to toxic concentrations of carbon tetrachloride in the air is productive of the following symptoms: nausea, vomiting, headache, dizziness, and irritation of the eyes, nose, and throat. These symptoms frequently disappear within a few hours after cessation of exposure. Absorption of larger quantities produces stupor, convulsions, coma, and death due to depression of the

central nervous system. Sudden death may occur from ventricular fibrillation or depression of the vital centers of the brain stem. After a delay of from several hours to 2 or 3 days, signs and symptoms of injury to the liver and kidneys may become evident. In some cases, initial symptoms of central nervous system depression are not followed by signs or symptoms of hepatic or renal damage. Conversely, hepatic and renal pathology may occur in the absence of any immediate effect on the central nervous system.

Common delayed findings include nausea, vomiting, abdominal pain, diarrhea, hematemesis, rapid enlargement of the liver, tenderness in the right upper quadrant, jaundice, and abnormal liver function tests. Wroblewski states that concentrations of glutamic oxalacetic transaminase in serum reach their highest values in viral hepatitis and carbon tetrachloride intoxication. Proteinuria, hematuria, and cylindruria precede anuria. Optic neuritis with decrease in the visual fields, necrosis of the adrenal cortex, and pancreatitis are infrequent complications. Pulmonary edema has been reported in some cases of carbon tetrachloride intoxication. This complication is probably a result of the combination of acute renal failure and damage to the myocardium and central nervous system; it may follow either ingestion or inhalation of the solvent. An attempt has been made to implicate phosgene as a cause of pulmonary edema in some of these cases. Thermal degradation of CCl_4 does result in the generation of phosgene. A study by Smyth and Smyth, however, demonstrated that no phosgene was produced when concentrations of up to 5000 ppm of carbon tetrachloride were present in the vicinity of a pilot light of a gas stove.

Exposure to 60,000 to 80,000 ppm for 30 to 60 minutes will cause either immediate or delayed death. Inhalation of 15,000 to 30,000 ppm for longer than one hour usually produces signs and symptoms of intoxication. The highest concentration which can be breathed for brief periods of time (one-half hour) without serious consequences is 1000 ppm.

Chronic Intoxication Due to Inhalation. Continued exposure to concentrations of carbon tetrachloride in excess of 100 ppm is productive of physiologic disturbances. Previous studies have shown that when carbon tetrachloride is present in levels from 100 to 300 ppm in the industrial environment, workers may suffer from nausea, vomiting, loss of appetite, abdominal pain, apathy or mental confusion, and loss of weight. Dermatitis has been described, but this is a consequence of the defatting effects of carbon tetrachloride on the skin.

Evidences of hepatic or renal damage by either clinical examination or laboratory tests may be present if the exposures are of sufficient magnitude and frequency.

A recent study of Kazantzis and Bomford reports symptoms of nausea and vomiting in 15 out of 18 workers exposed to between 45 and

100 ppm of carbon tetrachloride in air. The threshold limit recommended by the American Congress of Governmental and Industrial Hygienists is 25 ppm. The odor of carbon tetrachloride is detectable at 72 ppm. It is obvious, therefore, that the odor of carbon tetrachloride in an environment means that it is present in excess of the suggested limit for continued daily exposure.

Intoxication due to Ingestion. Ingestion of carbon tetrachloride results in a clinical picture similar to that seen in acute intoxication due to inhalation. Gastrointestinal symptoms are more severe with increased probability of hematemesis and abdominal pain. Depending upon the amount absorbed, hepatic and renal damage may become evident within several hours or may be delayed for 2 or three days.

The lethal dose by mouth of carbon tetrachloride is said to be as little as 2 to 4 ml for some susceptible persons.

Diagnosis of Carbon Tetrachloride Poisoning

The diagnosis of acute carbon tetrachloride poisoning is primarily dependent upon obtaining a history of the ingestion of the liquid or inhalation of its vapors. The history of the illness and progression of the clinical course should be compatible with that described above for carbon tetrachloride intoxication. There are no specific biochemical tests which serve to distinguish the effects of carbon tetrachloride on the liver and kidney from those of other poisons or infectious agents which exhibit similar hepatotoxic or nephrotoxic properties. The blood serum level of GOT is usually extremely high, but no higher than levels seen in infectious hepatitis. The identification of the chlorinated hydrocarbon in samples of blood, urine, or alveolar air substantiates a history of exposure to the solvent. In the absence of any quantitative data in the literature, however, such information is of little use in defining the degree of exposure.

At present, the clinical differentiation of chronic liver disease produced by carbon tetrachloride is an extremely difficult problem except in cases in which a liver biopsy demonstrates the presence of alcoholic hyalin, biliary cirrhosis, or other specific diseases. A decision as to whether repeated exposure to CCl_4 has caused permanent hepatocellular damage or aggravated pre-existing liver disease must rest upon: (1) careful medical evaluation of the patient to determine the presence of other factors (hemochromatosis, biliary cirrhosis, et cetera, (2) a definite history of (repeated) exposure to the agent, and (3) some information, if possible, on the range of concentrations of CCl_4 in the ambient air at the site of exposure.

Treatment of Carbon Tetrachloride Poisoning

The therapy of any case of poisoning should be oriented toward: (1) preventing further absorption of the material, (2) increasing the rate of the removal

of the poison from the body, (3) specific treatment measures directed at preventing the poison from accomplishing its toxicologic actions, and (4) supportive treatment for the patient who has suffered damage to tissues. With respect to carbon tetrachloride, it is usually impossible to prevent further absorption or enhance the rate of removal of carbon tetrachloride from the body. If carbon tetrachloride has been ingested, it should be removed by gastric intubation; sodium or magnesium sulfate, 15-30 gm, in water should be administered as a cathartic.

Supportive treatment for hepatic damage includes administration of certain agents which have been suggested in animal experimentation to be of protective value, but which have not proved to be equally effective in man. These include methionine and vitamin B_{12}. Calcium has been shown to have some protective and therapeutic benefit in animals and man. If there is no evidence of renal failure by the second or third day following exposure, a high-carbohydrate, high-protein diet should be instituted. Treatment of the hepatic injury must be modified by the thought that in all probability acute renal failure may be forthcoming. Details of management of anuric patients have been well outlined elsewhere. The presence of concomitant renal and hepatic failure makes the clinical situation a most difficult one.

The most effective treatment of carbon tetrachloride poisoning is to prevent its occurrence. Any use of carbon tetrachloride in industry should be carefully studied and other less toxic solvents substituted whenever possible. When its use is necessary, strict control of the environment must be maintained, and employees should be carefully screened to exclude from exposure those with a past history of liver or renal disease. Periodic clinical and laboratory examinations should be performed on exposed workmen.

Prevention of accidental poisoning in the home is a problem confronting all physicians. Any solution to this must include adequate labeling of all products containing carbon tetrachloride and education of the public concerning proper storage and use of chemicals in the home.

(NOTE: Carbon tetrachloride has been largely eliminated for use as a cleaning solvent in the Navy. Its limited usage is strictly controlled by the provisions of BuMed Instruction 6270.2 which requires that all requests for carbon tetrachloride should be reviewed by the commanding officer, and approval given only if no suitable substitute is available to meet the performance specification. Such instances are unusual. BuMed Instruction 6200.5 contains detailed information on the stringent safety precautions necessary for its use.)

* * * * * *

The Diabetic is Employable

Hugo T. Engelhardt MD and Harvey B. Snyder MD, Department of Medicine, Baylor University College of Medicine, Houston, Texas.
J Occup Med 2: 427-430, September 1960.

Diabetes mellitus is by no means a rare disease. It is estimated that almost 5,000,000 persons in the United States are either diabetics or potential diabetics. Almost 75% of these are more than 40 years of age. It is in this group that experienced and skilled employees may be found. Dr. Howard Root has stressed the fact that this makes diabetes mellitus an important disease in industry.

In a study of 500 persons chosen at random in a large industry, 2% were found to have definite evidence of the disease, while another 1.4% satisfied the criteria for potential diabetes. It is significant that the average age of those tested was 40 years.

In order to demonstrate the working experience of diabetics, office personnel of a large industry were studied. Twenty-three instances were discovered in which a definite history of diabetes could be determined. The average age of the subjects was 56.8 years; the duration of their disease averaged 8.52 years. To determine the average number of days lost from work by these persons, their work histories for the last 5 years were investigated. Their average number of days absent because of sickness was 6.94 per year. This compares with the figure of 4.61 days per year for the nondiabetic office personnel and 6.97 days per year for the same company's entire working force over the same 5-year period.

Because of the age of the subjects, many complications were observed. As one would expect, the cardiovascular system was affected in a number of instances. Two subjects had coronary artery disease exemplified by the anginal syndrome. Four had benign essential hypertension. Two subjects had evidence of peripheral vascular disease. Although gout is an uncommon complication of diabetes, it is of interest that 2 of the subjects suffered from a combination of these diseases. Diabetes of one person was complicated by bronchial asthma. This, like gout, is infrequently seen in persons exhibiting diabetes mellitus. One of the subjects, who had had diabetes mellitus for 10 years, had been absent from work 142 days over the past 5 years; it is significant that this case was complicated by alcoholism. Another subject in this study—diagnosed as a diabetic a little more than a year ago—had been absent from work 174 days in the past 5 years; this case was also complicated by alcoholism. The absence of these 2 employees totaled 316 days for the past 5 years or more than 39% of the total absenteeism of the 23 subjects. There were 2 persons whose only abnormality was marked obesity; 6 subjects were considered healthy.

The group was composed entirely of executives and white-collar workers; as a result, the accident experience was negligible. In their study, Weaver and Perret reported a disabling-injury rate of 15.6 per 1,000,000 hours of exposure compared to 12.9 for the nondiabetic group. They point out that these represented very few disabling injuries; hence, no great statistical value can be placed on them. Dublin and Marks indicate that the accident experience of diabetic workers is a satisfactory one. Their figures reveal that the frequency rate was 7.8 per 10,000 exposure hours for nondisabling injuries among diabetics, whereas for a control group, it was 7.4. They suggest the possibility that the diabetic workers were more conscientious in reporting even minor disabilities because of their knowledge of the role which infection plays in diabetes; this factor, the authors propose, might have influenced the reported rates.

It is of interest that all the authors' subjects were working in an unrestricted capacity at the time of the study. A number of the group have since retired; in no instance was retirement on a disability basis. All of these people have continued under the care of their family physicians with a little more than one-third requiring insulin in daily doses varying from 10 units of a long-acting insulin to 60 units of mixtures of protamine zinc and crystalline insulins. None has suffered hypoglycemia nor has there been an instance of diabetic coma. It must be emphasized that the type of diabetes exhibited by these persons is of the adult variety, and they have responded well to therapy.

Any physician in industry is aware of the discrimination which—rightly or wrongly—exists in the minds of those who are asked to hire a diabetic.

In a classic study in 1938, R. D. Lawrence and C. Matters observed that diabetics suffered great difficulties in their work and employment from the public misconception that they were really invalids. They expressed the opinion at the time that the attitude of government and industry was unjust and that it was a survival of the attitude common in preinsulin days when ill health and early death were common among diabetics. Unfortunately, 20 years later, this opinion is still widely held. The fact is that the life expectancy of diabetics has steadily increased.

It is difficult to get a clear picture of the life expectancy and mortality of diabetics in these times when new methods of treatment are being presented to the medical profession almost daily. A widely quoted study is that based on the experience of the Joslin Clinic for the 5-year period, 1947-1951. It must be remembered that these diabetics first came under observation during the years 1930 through 1951. These data show that the life expectancy of a 30-year old diabetic was approximately 30.1 years, while that of the general white population was 42.5 years. The mortality rate for the same period at age 30 was 15.2 per thousand diabetics compared with 1.5 for the general white population.

The impact of modern medicine on this disease has resulted in increased life expectancy. A diabetic who has had the disease for less than 10 years at age 30 should expect to live to age 65, whereas a nondiabetic may expect to reach age 71. The diabetic who has had the disease 10 to 20 years at age 30 should expect to live to age 63.

In recent years, studies have been made regarding the employability of diabetics because of their improved health and longer life expectancy. Soskin has posed two questions in this regard: (1) From the standpoint of productivity and absenteeism, do diabetics make good employees? (2) If they do, does the presence of diabetes in any way affect their capability to carry out the job? The authors' study is limited to those persons who developed diabetes during their years of employment. Their experience in industry and in large diabetic clinics prompts them to answer Soskin's questions in this way: In the executive and white-collar class, diabetics compare favorably with nondiabetics in terms of productivity and work attendance. The fact that these subjects have diabetes has no significant effect on their ability to do their work.

The statisticians, Dublin and Marks, in their study of diabetics in industry, conclude that the work record of diabetics is a satisfactory one and that the large majority of diabetics who are under good medical care can discharge their duties as well as nondiabetics. They emphasize—as did Lawrence—that even today the diabetic too often has difficulty obtaining and holding a job once the presence of his disease is known. They point out that this policy is due to the fact that the attitude of management has been colored either by unfavorable publicity or by experience with an exceptional case of diabetes. No one questions the fact that there are real problems with the diabetic, but many of these problems no longer have the importance which they once had: witness the ability to treat infections effectively, improvements in diet, refinements in insulins, and recently, the oral hypoglycemic agents.

The Committee on Employment of the American Diabetes Association has sifted through the information obtained from hundreds of companies employing diabetics. Their information indicates that the diabetic who is well controlled and well regulated and who keeps himself constantly under medical supervision is an acceptable employee. Moreover, because of his self-discipline, a diabetic often becomes an outstanding employee in whatever position he may occupy.

The Committee has formulated certain standards for the employment of diabetics. Among these is a request that the diabetic on seeking employment should bring with him a note from his physician stating how well his disease is controlled. The Committee feels that a diabetic is capable of performing any type of work for which he is physically, educationally, and mentally prepared.

Individuals who require large doses of insulin should not be assigned work in which the hypoglycemic attacks might be hazardous to

themselves or others. The chance discovery in 1955 by German investigators of the hypoglycemic effects of certain sulfonamides—BZ55(carbutamide) and its successors—might well be the key to this problem. Ideally, diabetics should have regular working hours and should avoid the shift from midnight to 8 a.m. This is the one concession that even well controlled diabetics should require. As oral hypoglycemic agents are discovered and improved, however, even this concession may be waived. Diabetics using insulin are considered controlled, according to the Committee, if the fasting blood sugar is not below normal limits nor above 150 mg/100 ml, Folin-Wu; if the 3-hour postprandial blood sugar is not higher than 250 mg/100 ml, Folin-Wu; and if the patient is under regular medical supervision. Should the diabetic be under observation by means of the 24-hour urine sample, he would be considered to be regulated if he excreted less than 10% of the total available carbohydrate in his diet in 24 hours.

The diabetic adult who requires no insulin to control his disease presents no problem to management. For purposes of employment, persons in this category may be considered in many instances in the same fashion as nondiabetics. At the other extreme is the uncooperative and poorly regulated diabetic job applicant whom every employer is justified in refusing employment. Such cases can usually be effectively screened out by preemployment interview and examination procedures. Between these two extremes there is a large group of diabetics who follow their physician's advice and understand their disease; as a result, they are well controlled. When these persons are given appropriate positions, they make good employees.

* * * * * *

Nightmare Hazards - Unusual Explosions

Technicians in the Experimental Fuze Unit of the Arsenal Operations Division at Picatinny Arsenal, Dover, N.J., work with hazards they cannot see—low-energy initiators which can be detonated by small electrical currents. The low-energy explosives are so sensitive to electromagnetic fields that they may be detonated by impulse from the generator of a passing car or even the static electricity created by a person combing his hair.

Safety experts have undertaken an elaborate safety program which greatly reduces the possibility of an accidental detonation. All light switches and bulb sockets are specially designed explosion-proof fixtures. Telephones are designed to prevent emanation of electric waves. The clothing of all workers is carefully monitored. Only conductive-soled shoes and cotton clothing are worn, because regular-soled shoes prevent drainage of static electricity and clothing of synthetic materials tends to create static electricity. Chemically-treated, flame-resistant uniforms with no pockets or cuffs which would collect explosive dust are provided.

Jewelry and combs are prohibited. When explosive items are removed from their protective aluminum foil wrappings to be packed into casings, technicians wear metal "wristlets" to which ground wires are attached. Visitors drag metal canes after them to drain off any static electricity.

Nothing is overlooked to protect the safety of those who work with sensitive explosives. (Anon., Safety Maintenance, December 1960; abstracted in Industr Hyg Dig, January 1961)

* * * * * *

Field Test for Air-Borne Lead

A field test is described (B.E. Dixon and P. Metson, Analyst, February 1960) for determining small amounts of total air-borne lead in industrial atmospheres; it can be carried out on the sample used for determining lead fume by the authors' staining method. The sample is collected on a test paper, which is then treated in a graduated sample tube with cold dilute nitric acid containing hydrogen peroxide to dissolve the lead dust and disintegrate the paper. Measured amounts of ammoniacal cyanide-metabisulfite-citrate solution of dithizone in carbon tetrachloride are successively added, and the lead dithizonate is extracted at pH 11. The color of the lower layer is compared with aqueous color standards or permanent glass color standards. (Industr Hyg Dig, January 1961)

* * * * * *

DEPARTMENT OF THE NAVY
U. S. NAVAL MEDICAL SCHOOL
NATIONAL NAVAL MEDICAL CENTER
BETHESDA 14, MARYLAND

OFFICIAL BUSINESS

Permit No. 1048

POSTAGE AND FEES PAID
NAVY DEPARTMENT